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Gastroesophageal Variceal Hemorrhage

Pathogenesis and Management

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. David W. Martin, Jr., Associate Professor of Medicine, and James L. Naughton, Assistant Professor of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, CA 94143.

DR. NAUGHTON:* *The topic for consideration today is variceal hemorrhage. We have asked Dr. John P. Cello, Chief of Clinical Gastroenterology of San Francisco General Hospital, to discuss the topic, with Dr. Robert Byers first presenting a case recently seen on the medical service.*

DR. BYERS:† The patient is a 62-year-old white woman with an approximately two-year history of hyperthyroidism. She was seen by her physician one year before admission here for epistaxis and palmar erythema. Hepatomegaly was noted together with abnormal findings on liver function tests. Results of a biopsy of the liver done at that time were interpreted as mild persistent hepatitis. The patient was treated with prednisone in a tapering dosage to 10 mg a day for the last four months. She was admitted to a local hospital for hematemesis and melena 1½ weeks before admission here; endoscopy at that time showed blood loss from varices. She was given a transfusion of 6 units of blood and sent home, but two days following discharge she returned to the local hospital with orthostatic hypotension and black stools. She was given another transfusion of 6 units of blood and was transferred to the University of California Medical Center. She had no history of alcohol abuse. She was well developed, well nourished and in no acute distress; she was alert and cooperative. Vital signs were normal and without orthostatic changes. Spider telangiectasias on the trunk and upper arms were noted, bibasilar rales were present on examination of the chest, and examination of the abdomen showed the liver to have a 12-cm span in the midclavicular line extending 2 cm below the right costal margin. A firm, tender hepatic edge was palpated. The spleen tip was felt. Ascites was not noted, but palmar erythema was obvious. All stool specimens were melenic.

Laboratory studies on admission disclosed the following values: leukocytes, 11,000 per cu mm; hematocrit, 29.2 percent; total bilirubin, 1.3 mg per dl; serum alkaline phosphatase, 38 units per dl; lactic dehydrogenase, 173 units per ml; serum glutamic oxaloacetic transaminase, 50 units per ml. Other laboratory values were normal and a study was negative for hepatitis B surface antigen. A radiograph of the chest showed an elevated right hemidiaphragm and small right pleural effusion.

The hospital course was marked by a drop in hematocrit to 25.7 percent and a transfusion of 1,000 ml was required. Endoscopy showed large esophageal varices without fresh blood loss. Some ascites was found on abdominal sonography. Examination of a biopsy specimen of the liver

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showed cirrhosis without hepatitis. The patient was transferred at that time to the surgical service, where a splenorenal shunt operation was carried out without complications. She was discharged and she returned home to the care of her private physician.

DR. CELLO:* A slightly more prosaic clinical description of a patient with portal hypertension was recorded in the 17th century. John Brown, Royal Physician at St. Thomas Hospital in London, was the first clinician to describe the appearance of the liver in a patient with portal hypertension and gross ascites.[1] In 1685 Brown wrote "I send you here the figure of a hydropical (that is, an ascitic) person. He was about 25 years of age, a soldier in one of His Majesty's regiments here in town who contracted his distemper by drinking much water, when he could not stir from his duties and catching cold at night in being upon the guard. His swellings resumed so there was nothing more now to be thought of but a paracentesis, whereby we drew from the patient three pints of brinish liquor and within three days as much more; the next day he died." Brown describes the appearance of the liver as follows: "The liver consisted in its concave, convex and inward parts of glands which made up the whole substance." However, it was René Laennec who first used the term "cirrhosis" in 1826 to describe the gross appearance of the liver in a patient with portal hypertension and ascites. He coined the word *cirrhosis,* from the Greek *scirrhus,* meaning of yellow or tawny color.[2]

There are several aspects of portal hypertension and variceal hemorrhage that should be considered. First, I shall review our understanding of the pathophysiology of portal hypertension and the development of gastroesophageal varices. Second, and quite distinct from the first (because we know that there can be varices in patients for years without blood loss), I shall consider the pathogenesis of the actual torrential hemorrhagic episode as a complication of varices. Third, some aspects of the medical and surgical management of esophageal varices will be reviewed.

Pathophysiology of Portal Hypertension

The liver begins an intimate relationship with portal venous blood (blood returning from the bowel) early in embryonic life when the ventral hepatic diverticulum develops from the foregut invading the omphalomesenteric vein. This embryonic vein receives blood from the yolk sac, a structure continuous with the primitive gut. The omphalomesenteric vein of the embryo eventually becomes the portal vein in adults.

With the body at rest, between digestive periods, total hepatic blood flow in an adult is 1,500 ml per minute, or roughly 58 ml per 100 grams of liver per minute. This represents 28 percent of the cardiac output and 20 percent of the total oxygen utilization of the body at rest. During digestion, arterial-portal venous oxygen differences are notably increased due to increasing utilization of oxygen in the intestine, and the increased oxygen demand by the liver during digestion is supplied by increasing hepatic arterial blood supply. Of the 1,500 ml per minute of total hepatic blood flow, 1,000 ml, or two thirds, is portal venous flow. In addition, portal venous blood supplies 50 percent to 60 percent of the total oxygen utilization of the liver at rest. This important portal flow is perfused at a central portal venous pressure of 10 to 15 cm of water. The portal venous blood delivery system, therefore, is a high volume, low resistance system.

In this high flow, low pressure system, any lesion in the portal venous delivery system may increase portal venous resistance and lead to portal hypertension. According to Dame Sherlock and many other investigators, one can conveniently classify portal hypertension on the basis of the site of increased portal venous resistance.[3] In extrahepatic portal hypertension, classically one is dealing with either portal venous occlusion in which splenic or main portal veins are thrombosed or increased splenic blood flow due to an arteriovenous malformation or even to massive splenomegaly. Intrahepatic presinusoidal portal hypertension will occur in cases of schistosomiasis, in congenital hepatic fibrosis or with a variety of portal zone-infiltrative processes that can notably increase portal venous resistance. Sinusoidal portal hypertension and postsinusoidal portal hypertension are seen in a variety of processes such as cirrhosis and veno-occlusive disease of the liver.

Ordinarily the most common cause in the development of hypertension in our society is alcoholic liver disease (Table 1). Actually at a variety of stages in the development of alcoholic liver disease, one can find increasing portal venous resistance. Cirrhosis, however, is the stage

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TABLE 1.—*Causes of Portal Hypertension in 100 Consecutive Patients Admitted to the University of California Medical Center, San Francisco, and Affiliated Hospitals, for Portal-Systemic Shunts, 1974-1978*

Alcoholic Liver Disease	
Active alcoholic	75
Past alcoholic	10
HB_sAg^- macronodular cirrhosis	7
HB_sAg^+ macronodular cirrhosis	2
Hemochromatosis	2
Schistosomiasis	2
Wilson disease	1
Portal vein thrombosis	1
TOTAL	100

HB_sAg = hepatitis B surface antigen

in which there is a substantial irreversible component of portal hypertenson. The most important component of portal hypertension in the cirrhotic liver is the regenerative nodule.[4] Several factors are responsible for this.[5] The nodule is supplied almost exclusively by the hepatic arterial system. New portal venous tributaries are not grown in the hepatic tissue in the nodule. As the nodule grows and enlarges, it will first compress hepatic venous structures. Therefore, the nodule tends to produce a postsinusoidal portal hypertension. In addition, the nodule by its growth and enlargement will compress and distort the remaining normal parenchyma perfused by the portal vein. Consequently, increased sinusoidal resistance to portal flow occurs. A third factor in the development of portal hypertension in nodular regeneration is the formation of small hepatic arterial-to-portal-venous shunts in the broad bands of scar tissue between nodules.

Varices—One of Several Shunts

Whatever factors are responsible in portal hypertension—be they presinusoidal, sinusoidal or postsinusoidal, alcoholic liver disease, or postnecrotic cirrhosis—the most clinically notable sequela of cirrhosis, other than hepatic failure, is the development of a large number of portal-venous to systemic-venous shunts. The most clinically significant shunts are those that occur at the gastroesophageal junction (producing varices), the umbilicus (producing the caput medusae) and the rectum (as large internal hemorrhoids). Other clinically insignificant but occasionally prominent shunts occur around the diaphragm and around the spleen.

The most important of these shunts are those

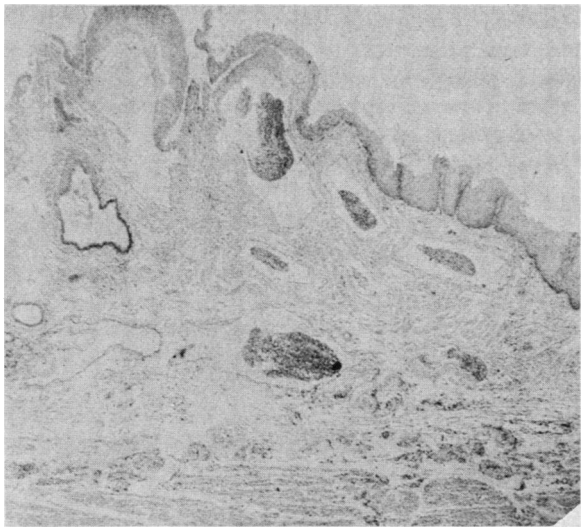

Figure 1.—Esophageal varices. Thin-walled venous channels are noted in the submucosal loose connective tissue. Only the stratified squamous epithelium of the esophagus separates the portal blood from the lumen.

that develop at the gastroesophageal junction, where, through increased resistance to portal flow, a large number of potential venous channels open. These channels, in fact, are simply the submucosal venous channels (the venous plexus) that are ordinarily found in the submucosa of the tubular gastrointestinal tract. Under the influence of portal hypertension, portal venous flow is channelled from a relatively high resistance region in the liver to the relatively low resistance system that is adjacent to the esophagus; namely, the azygous and hemiazygous systemic-venous systems. These variceal venous channels, therefore, are not newly formed, but rather massively dilated, submucosal venous channels made up of extremely thin-walled vessels that are precariously approximated to the lumen of the esophagus (Figure 1). Endoscopically, varices appear as large, soft, fleshy and tubular nodular masses that run longitudinally down the esophagus. In some patients the varices are so prominent that they touch each other across the lumen and may almost completely occlude the lumen.

Varices, therefore, commonly develop in patients with portal hypertension. However, we know that there can be portal hypertension and varices in patients for years without blood loss. Moreover, there can be hemorrhage from varices that is followed by a relatively quiescent period of many months. Because variceal hemorrhage is the most life-threatening consequence of cirrhosis,

TABLE 2.—*Pathogenesis of Variceal Hemorrhage in Cases of Portal Hypertension*

Site—esophagus (from 20 to 40 cm from incisors); stomach: gastric varices high in cardia or fundus
Pathologic condition—small punctum or erosion; no esophagitis, ulcer or laceration
Factors responsible—unknown/undocumented • hypotension of lower esophageal sphincter and reflux esophagitis • Mallory-Weiss tears over a varix • esophageal/gastric peptic ulceration • surging increases in portal-pressure Valsalva maneuvers, straining, coughing, increasing ascites

what can be understood about the factors responsible for hemorrhagic episodes?

We know from studies of patients and epidemiology that the site of blood loss is invariably in the distal esophagus or proximal stomach (see Table 2); consequently, the proper term to describe this blood loss would be gastroesophageal variceal hemorrhage. The pathologic condition at the time of endoscopy or postmortem examination is a punctum or small isolated erosion. It is uncommon to see esophagitis, an ulcer or a laceration. What factor is responsible? There are a variety of theories that have been proposed to explain the development of the actual bleeding episodes. Is there hypotension of the lower esophageal sphincter that allows reflux of gastric acid and reflux of esophagitis overlying the varix? Is there a Mallory-Weiss tear, a longitudinal linear tear in the mucosa overlying the varix? Is it peptic ulcer? The factors responsible are not defined but some of these questions can be answered. In spite of the massive ascites possible, there is no demonstrable hypotension of the lower esophageal sphincter in those patients in whom studies have been done shortly after an exsanguinating variceal blood loss. Before and after diuresis, the mean pressure of the lower esophageal sphincter in these patients is normal.[6] This is further supported by our endoscopic and pathologic correlations. Esophagitis is not commonly found on endoscopy in the region of a varix from which there is blood loss. Moreover, rarely do we encounter Mallory-Weiss lacerations in the region of variceal hemorrhage. Peptic ulceration is also not commonly found. However, we know that there is no blood loss from varices unless intravenous pressures rise to dramatic levels, possibly associated with straining, coughing or Valsalva maneuvers. In addition it appears that exsanguinations requiring control through surgical therapy are unlikely to occur unless the portal venous pressures surge above 45 to 50 cm of water.

Diagnosis

Given our understanding of the pathogenesis of portal hypertension and variceal hemorrhage, how should these conditions be diagnosed? Is there any way one can suspect, from a patient's presentation, that an episode of blood loss is variceal in nature? Usually variceal hemorrhage is sudden and voluminous with hematemetic episodes unassociated with epigastric pain. On occasion, hemorrhage is less dramatic, such as was seen in the case under discussion, in which several days of painless melena were noted. The latter aspect of variceal hemorrhage was generally not appreciated until endoscopy was carried out routinely and we noted large eroded varices in patients presenting with several days of melena. On physical examination, as exemplified in this case, portal hypertension may or may not be suggested. Patients with variceal hemorrhage may have *no* clinically detectable ascites, or other signs of cirrhosis or portal hypertension. In addition, on physical examination the finding of features suggestive of chronic liver disease and portal hypertension *does not* substantiate varices as the cause of hemorrhage. No laboratory test is specific for cirrhosis and portal hypertension, and even standard single-contrast upper gastrointestinal series may fail to detect varices radiographically in 60 percent of the cases. Moreover, detection of gastroesophageal varices on barium studies does not confirm them as the site of hemorrhage.

Endoscopy is the only practical method for confirming varices as the site of upper gastrointestinal hemorrhage. The ideal time for carrying out this examination is shortly after a patient has arrived at the hospital and his condition has been stabilized. Large blood clots that will obscure vision must be evacuated from the stomach. When the stomach is clean, fresh blood loss from a punctate area overlying a varix, or an adherent fresh clot on a varix, confirms the site of hemorrhage.

Initial Medical Therapy

Variceal hemorrhage is not a minor problem in San Francisco. Every year 30 to 50 patients with variceal blood loss are admitted to San Francisco General Hospital (Table 3) and 14

TABLE 3.—*Causes of Significant Upper Gastrointestinal Hemorrhage, San Francisco General Hospital (100 consecutive cases)*

Lesion	*Documented by Endoscopy*
Esophagitis	4
Esophageal cancer	1
Esophageal varices	14
Mallory-Weiss tear	13
Total esophagus	32
Gastric ulcer	20
Gastritis	14
Gastric cancer	3
Pyloric channel ulcer	6
Total stomach	43
Duodenal ulcer	22
Duodenitis	3
Total duodenum	25
TOTAL	100

percent of the episodes of significant upper gastrointestinal hemorrhage occur in varices. The latter figure is verified in other series in urban populatons.

What should be the approach in cases in which blood loss is thought to be from varices, particularly in patients with florid stigmata of chronic liver disease? Obviously resuscitation is the first step; little can be done or should be done diagnostically unless the patient's condition is first hemodynamically stabilized. Certainly in many cases in which there is blood loss from varices, several units of blood are lost and prompt fluid replacement is required. However, with the attempt to restore intravascular volume, many patients who have had blood loss from varices are overhydrated or hypertransfused. In principle this would not appear to be hazardous in these patients. However, some experimental data suggest that there is a unique intravascular volume-portal pressure relationship in patients with cirrhosis, particularly in those with ascites in whom the ability to accommodate excess amounts of fluids is limited; this is manifested by surging increases in portal pressure in patients with volume overload.[7,8] Because a patient with ascites and cirrhosis is unable to accommodate extraordinary amounts of volume replacement, it is generally recommended that the central venous pressure be kept below 5 cm of water. In elderly patients or patients with cardiopulmonary disease, the central venous pressure may not accurately

TABLE 4.—*Proper Usage of Vasopressin (Pitressin) for Variceal Hemorrhage*

Preparation: use only aqueous vasopressin, not oil.
Route: intravenous continuous infusion
no need for intraarterial dose
Dosage: range for 70-kg man—
0.1 to 0.4 units per minute.
no loading dose
no tapering needed
Patient location: intensive care environment
Major side effects: depressed cardiac output,
hypertension,
coronary ischemia

monitor left-heart filling pressures and may incorrectly estimate intravascular volumes. In these circumstances thought should be given to placing a Swan-Ganz pulmonary artery catheter.

In many cases in which hemorrhage is great, vomiting and water lavage may contribute to pulmonary aspiration. It may be necessary to place an endotracheal airway to preclude aspiration. Elective intubation of a patient may be of additional benefit. Because many of these patients are agitated, uncooperative and alcoholic, diagnostic endoscopy and even placement of a balloon tamponade will be facilitated by airway intubation and muscular paralysis induced by pancuronium bromide or succinylcholine chloride.

Vasopressin Therapy

Vasopressin therapy is effective in moderating severe hemorrhage from the gut. The site of action of vasopressin infusion in patients with variceal blood loss is the splanchnic arteriolar bed, and not the varix. There is no vasopressin-responsive vascular smooth muscle in the portal venous system. At the level of the splanchnic arteriole, a relatively nonspecific vasoconstriction occurs with vasopressin therapy. This splanchnic oligemia induced by vasopressin causes a notable decrease in portal venous flow and pressure. There is no effect of vasopressin infusion on the hepatic artery. In fact, in some recent studies it is documented that with the decrease in portal venous flow, the hepatic artery alone in the splanchnic bed actually vasodilates, maintaining near normal total hepatic blood flow.

How should one administer vasopressin (Table 4) to a patient with variceal hemorrhage? Results of studies using rhesus monkeys, show that there is a stepwise decrease in superior mesenteric arterial blood flow, and a concomitant decrease in portal venous pressure as one increases vasopressin infusion logarithmically.[9] Earlier clinical experience

suggested that selective arterial infusion of vasopressin into the left gastric artery was necessary. The efficacy of selective intraarterial vasopressin infusions has been shown in recently published primate studies employing electromagnetic flow meters and manometers in the superior mesenteric artery and portal veins. The intraarterial infusion of 5 mU of vasopressin per kg of body weight per minute results in a prompt decrease of the arterial blood flow and portal venous flow, and a profound drop in portal venous pressure. At this low dose—equivalent to 0.35 units per minute infusion of vasopressin in a man weighing 70 kg (154.3 pounds)—systemic side effects could be shown with an elevation of central aortic blood pressure and a decrease in cardiac output.[9] Thus, continuous intraarterial vasopressin therapy, while effective in decreasing portal flow and pressure, does have undesirable systemic side effects (Figure 2A).

With continuous systemic intravenous vasopressin infusion at the same dose in primates (5 mU per kg of body weight per minute), comparable effects on splanchnic arterial flow and portal pressures are shown. A prompt decrease in superior mesenteric arterial blood flow, an increase in peripheral vascular resistance, and a prompt decrease in portal venous flow and pressure occur with intravenous infusions (Figure 2B). Systemic side effects are comparable whether the dose of vasopressin is administered intraarterially or intravenously.[10,11]

Our recommendation, based on these and other studies and on clinical experience, is that the vasopressin be administered by continuous intravenous infusion at a dosage of approximately 5 mU per kg of body weight per minute. Thus, we would recommend administering between 0.1 and 0.4 units per minute in an adult, as a continuous intravenous infusion. The onset of action is so prompt that no loading dose is necessary, and no tapering is needed when administration of the drug is discontinued. Another important clinical point is that toxicity of vasopressin is definitely related to systemic blood levels. The drug has extraordinary toxicity in some patients with hypertension, low cardiac output and coronary ischemia shown by ischemic changes on an electrocardiogram. Therefore a patient's condition should be closely monitored while vasopressin is being administered. The toxicity is relatively unpredictable. In some elderly patients a full effective dose of vasopressin infusion may be administered without any problems, while in some younger patients significant coronary ischemia occurs.

Balloon Tamponade

Sengstaken-Blakemore and Linton tubes have been used for years in patients with massive blood loss from varices. So much morbidity and mortality has been associated with the use of balloon tamponade therapy in cases of hemorrhage from esophageal varices that, in some centers, the use of balloon tamponade has been abandoned in favor of vasopressin infusion or emergency surgical operation. Our experience at hospitals affiliated with the University of California, San

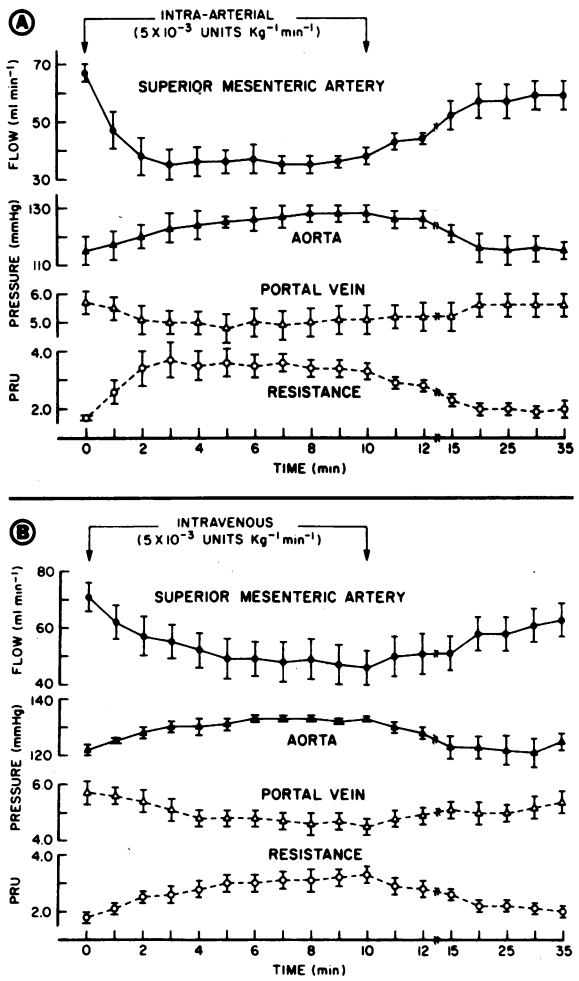

Figure 2.—Vasopressin administered to five monkeys intraarterially **(A)** or to six monkeys intravenously **(B)**. The infusions begin at minute 0 and continue for ten minutes. Superior mesenteric artery blood flow fall in both situations within a few minutes of infusion. (Reproduced with permission from Freedman AR, and *Gastroenterology.*[9])

TABLE 5.—*Suggested Physician Orders for Maintenance of Minnesota Balloon Tamponade*

1. Chest radiograph, portable, to document subdiaphragmatic location of gastric balloon.
2. Keep gastric balloon inflated with 400 ml air; double-clamp inflation port; *do not deflate.*
3. Apply tension to balloon by taping insertion tube to corner of mouth, using crossed wooden tongue blades, cushioning mouth with gauze.
4. Change tube position from one corner of the mouth to the other every four hours.
5. Esophageal suction port—intermittent suction; *never lavage via this port.*
6. Gastric suction port—maintain on low intermittent suction; may lavage and administer antacids/lactulose via this port.
7. Esophageal balloon (if necessary)—inflate with air monitoring pressure using aneroid manometer. Never inflate above 40 mm of mercury. Deflate esophageal balloon for 30 minutes every six hours.
8. Keep patient without oral intake.
9. Elevate head of bed 15 degrees.
10. Encourage good mouth care and frequent deep breathing.
11. Tape scissors near bedside. If respiratory distress caused by tube, cut entire tube assembly and remove immediately.

Francisco, Medical Center has been positive. The tamponade is effective in arresting torrential hemorrhage from varices and allows hemodynamic stabilization in almost all cases, even in those ultimately requiring emergency surgical therapy . These favorable results are explained by our restricting the insertion and maintenance of tamponade to patients who have endoscopically proven blood loss from varices, and by requiring that the procedure be done by a senior gastroenterology or surgical resident. The most common cause of morbidity associated with the use of balloon tamponade in other series is aspiration pneumonia. Several advances in the design and construction of newer balloons have helped make this complication less likely. The Minnesota tube, the balloon tube now in use at the three hospitals affiliated with the University of California, San Francisco, has incorporated into the insertion tube, a separate lumen for aspirating esophageal secretions. With Linton or Sengstaken-Blakemore balloons inflated, occluding the gastroesophageal junction, pharyngoesophageal secretions and blood could pool in the esophagus and spill over into the trachea. The esophageal suction ports in the Minnesota tube, however, can prevent this and, therefore, we believe that it is the instrument of choice for maintaining balloon tamponade. To maintain effective airway cleansing in patients with vigorous hemorrhage from esophageal varices, intubation is carried out before the insertion of tamponade balloons. Strict orders should be written and carried out for care of patients receiving balloon tamponade therapy (see Table 5). Another interesting aspect of the new Minnesota balloon has been successful tamponade of esophageal varices through the use of gastric balloons alone. By inflating a gastric balloon with 400 ml of air and applying gentle traction to the mouth, the gastric fundic varices supplying blood to the esophageal varices are compressed and esophageal balloons almost never have to be used. The latter aspect of the balloon tamponade, esophageal balloon inflation, is the most difficult to maintain and has been associated with esophageal ulceration and perforation. In our experience, the large gastric balloon of the Minnesota tube has been highly effective alone in arresting variceal hemorrhage.

Balloon tamponade is a temporizing procedure, but one that allows for more careful preparation of patients for surgical operation. In 85 percent of patients in whom balloon tamponade is used, hemorrhage will be controlled. However, in a small percentage of patients, balloon tamponade is ineffective. Following deflation of the balloon, blood loss will occur again in a high percentage of patients, necessitating reinsertion of the tube. Tamponade with esophagogastric balloons is an effective hemostatic maneuver, and not the primary therapy for hemorrhaging from varices. However, in our experience it is unusual for varices to continue bleeding in spite of the use of vasopressin alone, or in combination with balloon tamponade.

Surgical Therapy for Variceal Hemorrhage

Variceal hemorrhage is often severe, occurs and recurs unpredictably, and is difficult to treat quickly. Because of these factors, surgical decompressive procedures have been devised to reduce portal hypertension. Originally called Esk fistula, portal-systemic shunts have been developed over the years and include a variety of innovative decompressive procedures. The most commonly used are end-to-side and side-to-side portacaval shunts, and splenorenal shunts. In patients with large cirrhotic livers, a variation of the side-to-side portacaval shunt can be constructed by using a graft of Dacron or autogenous jugular vein. This "H-graft" or Drapanas mesocaval shunt is often used at San Francisco General Hospital

TABLE 6.—*A Partial Listing of Factors Showing Association With Favorable Outcome Following Portal-Systemic Shunts*

Favorable Survival Factors*
Younger patients
Good nutritional status
Good muscle mass
Nonencephalopathic before shunt
No notable ascites
Nonalcoholic liver disease
Abstaining from alcohol
No chronic active hepatitis or alcoholic hyaline on biopsy
Albumin >3.0 grams per dl
Bilirubin <2.0 mg per dl
Prothrombin < two seconds prolonged
Serum glutamic oxaloacetic transaminase <100

*Composite series from multiple sources—not universally agreed upon by all investigators.

TABLE 7.—*The Child's Classification, Widely Used by Clinicians in Assessing Heptatic Functional Reserve in Patients With Cirrhosis*

	Status		
Test	*A*	*B*	*C*
Bilirubin (mg per dl)	<2	2-3	>3.0
Albumin (grams per dl)	>3.5	3.0-3.5	<3.0
Ascites	None	Easily controlled	Pronounced
Neurologic status	Alert	Early encephalopathic state	Coma

in patients in unstable condition.[12] Clotting in the prosthetic Dacron has been a problem in our series of patients, and this problem has been noted by others. When an internal jugular vein is used, autogenous Drapanas shunts are well tolerated without thrombotic complications. In many patients at Moffitt Hospital in San Francisco, retroperitoneal renosplenal or adrenosplenal shunts are carried out; these procedures require considerable surgical expertise but have the advantage of being constructed outside the ascitic peritoneal cavity. Our experience with these shunts has been excellent.

What are the indications for portal-systemic shunts? In whom should they be used? In patients with documented varices, and in whom there has never been blood loss from varices, shunts should not be used. Although there is excellent evidence that variceal hemorrhage is avoided through the use of prophylactic shunts, decreased mortality from variceal hemorrhage in these cases is offset by the operative mortality and excess mortality from hepatocellular failure. Thus prophylactic portal-systemic shunts do not substantively improve long-term survival in patients with no blood loss from varices.[13,14]

Therapeutic portal-systemic shunts surgically created in patients who have had blood loss, or are having blood loss, from varices will arrest and prevent variceal hemorrhage. Recurrent variceal hemorrhage is unlikely in those patients in whom a technically adequate portal decompressive procedure has been carried out. However, when considering all patients with blood loss from varices, portal-systemic shunts will not improve long-term survival when compared with medical treatment.[15] In general, when therapeutic shunts are used, it appears that hepatic failure and operative mortality offset the improved mortality from hemorrhage that would be likely to accrue with patients having undergone successful portal decompression.[16]

A variety of factors have been examined to help predict outcomes of therapeutic decompression procedures and to select those patients likely to benefit from shunting. In spite of years of hemodynamic study, no predictive value has been documented in selecting patients or shunts on the basis of a variety of factors including portal blood flow, hepatic artery flow, hepatic artery/portal flow ratio, portal pressures or "maximum perfusion pressures."[17] A number of clinical factors, however, are associated with improved outcome (Table 6). We believe that Child's classification remains the most important determinant of outcome of surgical procedure (Table 7). Patients with a preoperative Child's classification of *A* or *B* have notably better chances of surviving surgical operation, and considerably better five-year and ten-year survival, compared with patients with Child's classification *C*.[18-20] Nonetheless, even in patients with Child's classification *C*, portal-systemic shunts, though they may not improve five-year survival, will prevent recurrent variceal hemorrhage. Our recommendations at San Francisco General Hospital are that in cases of Child's classification *A* or *B*, in patients with documented significant blood loss from varices (greater than 3,000 ml of blood transfusion), therapeutic portal-systemic decompression should be carried out when the patient's condition is hemodynamically stabilized. For patients with Child's classification *C*, in cases of acites, jaundice, malnourishment or encephalopathy, we recommend vasopressin therapy, blood transfusions and balloon tamponade to control hemorrhage. We do not advise surgical procedures for these patients unless all these other approaches

are unsuccessful in controlling blood loss or unless, with continued medical management, the patients improve neurologically and metabolically.

Variceal hemorrhage is common in our urban environment, especially in cases of chronic alcoholism. The medical management is temporizing at best; surgical therapy seems to improve long-term survival in only a few patients with good hepatocellular function. It appears that the best therapy is to prevent the most important cause of cirrhosis and portal hypertension in our society —alcohol abuse.

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Electrocoagulation: Making a Curable Cancer Incurable?

IT REALLY IS AMAZING to see how well these patients have done by the electrocoagulation technique, because the method really destroys all our principles of surgical therapy for cancer. For example, if the cancer is confined within the wall of the bowel then one electrodesiccates, goes through, the wall of the bowel into the fat and the fat protrudes, the natural boundary has been broken and, in a few instances, the patient then has repeated attacks of bleeding or of infections. And, a curable cancer has eventually been made incurable by the procedure.

—CLAUDE E. WELCH, MD, *Boston*

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